



Seminar

Contribution of corticothalamic dysfunction to network hyperexcitability and cognitive deficits in Alzheimer's disease mice

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Alzheimer's disease (AD) clinically presents primarily with progressive cognitive decline, and is also associated with an increased incidence of seizures. How network dysfunction and seizures might contribute to AD-related cognitive deficits is unknown. To investigate the potential mechanisms, we studied a transgenic mouse model that overexpresses mutant human amyloid precursor protein (APP), resulting in high levels of Aβ production. These APP mice develop epileptic spikes and convulsive seizures, as well as nonconvulsive seizures that suggest that the corticothalamic network may be involved. Our study suggests that corticothalamic dysfunction may be a common denominator in AD pathophysiology.

Tuesday, Sep 30th 2014

11:30 AM (Tea/Coffee at 11:15 AM)

Seminar Hall, TCIS